

Insecticides: Why are animals killed only some of the time?

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Abstract

Considerable effort has been devoted to the development of predictive models, based on the toxicity and other characteristics of pesticides, to anticipate their hazards in the field. It is doubtful that these models can predict the ecological effects of pesticides in the diverse settings where they will be used. Numerous inconsistencies exist in the persistence and biological effects of insecticides when they are used in different environments. Such findings should have been anticipated as ecological conditions vary among areas treated with insecticides. The fate of insecticides, their bioactivity, and their effects on animals are determined by specific physiochemical and biological factors that differ between environments. Examples of variations in insecticide effects under different ecological conditions are discussed.

Introduction

Insecticides seldom kill vertebrates every time they are applied. Many that cause periodic mortality can be used at other times without apparent losses of wildlife.

Application techniques, environmental factors, and the ecological and physiological susceptibility of species that are present all contribute to the relative hazards of insecticides in different areas. Registrations of the more hazardous insecticides that consistently caused adverse effects have usually been cancelled. However, this trial and error process to identify hazardous pesticides takes time and can result in serious wildlife mortality while registrations are being withdrawn. Increasingly, efforts are being made to develop models using laboratory-generated data from toxicity testing, QSARS (quantitative structure-activity relationships), and microcosm and simulation studies to predict hazards of pesticides before they are registered and placed in widespread use (Peterle 1991:150-156, Kendall and Lacher 1994, Fisher et al. 1993, Van Leeuwen and Joop 1993). I discuss some examples of variations in pesticide persistence, behaviour, and effects that make me

doubt predictive models for ecological risk assessment can be of much value.

Unexpected persistence of residues

Parathion on peaches

In the early 1960s, agronomists in California gathered data to show that parathion was effective against a pest damaging peaches. Rates required for control of the pest left residues on peaches that were below tolerance at harvest. With this information, California registered parathion for use on peaches. During the first year parathion was used, many workers harvesting the peach crop became ill. They were found to be debilitated by cholinesterase (ChE) inhibition from parathion exposure because high residues remained on the leaves and skin contact made the workers ill. This parathion episode illustrated that insecticide residues can persist for different lengths of time, depending on the specific materials on which they are deposited.

Toxaphene in Oregon lakes

A study by Terriere et al. (1966) provided an additional example of variations in pesticide persistence and effects. Toxaphene was

applied to two lakes in the Oregon Cascades to kill undesirable fish before restocking the lakes with trout. Toxaphene was applied in 1958 at a level of 40 ppb in Miller Lake, and in 1961 at 88 ppb in Davis Lake. Toxaphene residues measured in 1962 were found to be much higher in Miller Lake although it had been treated 3 years earlier and at one-half the rate as Davis Lake (Table 1). Fish were successfully planted in Davis Lake in 1962, 1 year after treatment with toxaphene. Introduced fish died in Miller Lake for at least 5 years after it had been treated. Miller Lake was deeper than Davis Lake, had a slower rate of dilution because of smaller inlet streams, and was less active biologically with fewer and less abundant organisms. In this case, it was the variation in the characteristics of the treated environments that resulted in toxaphene persistence and chronic mortality of trout in one lake and not the other.

DDT persistence in different environments
Crick (1990) gave the half-life of DDT in tropical soils as 1 to 4 mo. He reported that 70 kg/ha of DDT was applied to cowpeas near Ibadan, Nigeria, over a 4-year period. After the fourth year, only 2.8% of DDT applied remained in the soil. DDT applied for tsetse fly control in woodlands of Zimbabwe also had a half-life of only 90 to 125 days in different soil types (Grant 1993). In this semiarid savannah, disappearance of DDT was much more rapid than was found by studies conducted in more temperate regions at higher latitudes.

In the United States, high rates (20-50 kg/ha) of DDT were annually applied to fruit orchards through the 1960s. Cooke and Stringer (1982) estimated the half-life of DDT and metabolites in orchard soils they studied to be between 3 and 11 years. Kuhr et al. (1974) found one-half of the technical DDT applied to New York orchards between 1949 and 1960 still persisted in soil as metabolites in the 1970s, and even herbaceous plants were contaminated with residues as high as 7.7 ppm. The lengthy persistence of high DDT residues in orchards has been suggested as a possible source of DDE that continues to cause eggshell thinning in certain species of North American birds (Blus et al. 1987). The unexpected persistence of DDT in agricultural soils of the Yakima Valley, Washington, was reported by Johnson et al. (1988); they also reviewed findings of long-term persistence of DDT in California, Puget Sound, Wisconsin, Texas, and New Mexico.

These studies in different locations and under varying environmental conditions show it is impossible to define a predictable half-life for DDT. The same problem exists for all pesticides that become environmental contaminants; under certain conditions even organophosphate insecticides such as fenitrothion can persist in the environment for 1 year or more (Yule and Duffy 1972). If persistence cannot be reliably predicted, it becomes difficult to assess the period of pesticide hazard to the environment.

Table 1. Toxaphene residues in Oregon lakes treated for fish control.*

Sample	Davis Lake (Treated 1961 at 88 ppb)			Miller Lake (Treated 1958 at 40 ppb)		
	1962	1963	1964	1962	1963	1964
Water (ppb)	0.63	0.41	<0.2	2.10	1.20	0.84
Aquatic plants (ppm)	0.39	0.21	-	4.59	4.59	5.77
Aquatic insects (ppm)	1.43	4.47	-	2.67	2.67	-
Fishes	4.22	5.48	2.60	-	-	12.50

* Source: Terriere et al. (1966).

Unanticipated effects of pesticides

Avian eggshell thinning

It has been thoroughly demonstrated that DDE can make eggshells thinner in certain species of birds (Cooke 1973, Stickel 1973). When insecticides can cause these kinds of unanticipated effects, it seems rather pointless to suggest that predictive models can be used to prevent such ecological catastrophes in the future. We simply do not know enough about the toxic effects of pesticides or about the diverse physiologies and life history strategies of wildlife. For instance, DDE does not thin the eggshells of all avian species to the same degree and these differences may be due to the way species use their energy and nutrient uptake to make their eggs. Indeterminate layers, such as chickens, use the fats and nutrients ingested each day to make their daily eggs and DDE does not seriously thin their eggs. The levels of DDE in their diet each day most often would be quite low. Conversely, determinate layers, such as brown pelicans, often do not feed during courtship, nest building, and egg laying, activities that can extend over 1-2 weeks. The fats and nutrients used for their eggs must come from reserves they have stored before beginning to breed. It may be the high levels of DDE stored in fat

and released during egg formation that are responsible for eggshell thinning in predatory birds (but, see Anderson et al. 1969:105).

Indirect effects

Indirect effects are those that influence animals through changes in their environments caused by pesticides. Insecticides can reduce food availability for insectivorous animals. Herbicides kill plants and can reduce food and cover required by animals. In one study (Keith et al. 1959), applications of 2,4-D to mountain grasslands in Colorado decreased production of forbs, decreased the proportion of forbs in the diet of pocket gophers, and severely reduced the abundance of pocket gophers (Table 2). The fact that indirect effects of pesticides will occur is predictable and the general kinds of effects to be expected can be anticipated. Still, it is impossible to predict which chemicals will cause problems, where they will occur, and which species will be affected. Again it would appear that the intricate wildlife debilities caused by indirect and unanticipated effects can be identified only through field study after pesticides have been applied.

Table 2. Effects of 2,4-D on foods, diet, and abundance of pocket gophers in Colorado.*

Feature	Plots	
	Untreated	Treated
Forb Production (lbs/ac)		
Pretreatment	652	400
Posttreatment	537	119
Forbs in Diet (%)		
Pretreatment	82	82
Posttreatment	89	49
Abundance (#)		
Pretreatment	101	117
Posttreatment	110	15

* Source: Keith et al. (1959).

Variations in effects with application methods

Ultra low volume (ULV) formulations

In the 1960s, aqueous formulations of fenitrothion applied at 0.11 kg/ha were widely used for mosquito control in California. These treatments were not known to cause wildlife mortality. However, in the mid-1960s, ULV applications were introduced as a means of increasing the area that could be treated aerially with each tank of spray. Aqueous formulations required applications of up to 9.4 L/ha of spray, while ULV formulations could be applied at 118-177 mL/ha. Following introduction of ULV applications of fenitrothion, avian mortalities became common in California and have continued to be reported periodically. ULV applications result in smaller droplet sizes, more complete and uniform deposits on biological material in the environment, and a longer persistence of residues (Keith 1968). Likewise, Busby et al. (1991) found ultra, ultra low volume (UULV) sprays of fenitrothion increased cholinesterase inhibition and mortality in songbirds. They concluded that choice of application methods could make treatments of 140 g/ha of fenitrothion as hazardous as those at 210 g/ha. In addition, they felt fenitrothion hazards were not necessarily avoided by registrations based

only on application rates. The lesson here is that ULV applications can be more hazardous to wildlife than higher volume applications of aqueous formulations.

Type of aircraft

Experience of many entomologists and wildlife biologists has suggested that the kind of aircraft used to apply insecticides can influence the intensity of effects on both insects and vertebrates. Biologists seldom have a chance to assess the relationship between aircraft type and wildlife hazards. Peakall and Bart (1983, Table 13) presented information that allowed a comparison of fenitrothion effects on birds when applied by either TBM or DC-6 aircraft. Bird abundance was measured before and after fenitrothion applications of 420 g/ha to forest habitats in Canada. Bird abundance was consistently lower following TBM applications (Table 3). Busby et al. (1991) also concluded that aircraft size can influence exposure of songbirds to fenitrothion, perhaps through the relative penetration of small droplets into forest canopy.

Treatment strategies

Most treatments to control insects in crops, rangeland, and forests are made by applying insecticides in aerial swaths across the infested area until it has all been treated. However, in most grasshopper and locust

Table 3. Effects of fenitrothion on bird abundance in relation to the type of aircraft used to apply spray.*

Species	Type of aircraft	
	TBM	DC-6
Northern parula (<i>Parula americana</i>)	-53	-8
Black-throated green warbler (<i>Dendroica virens</i>)	-56	-23
Yellow-rumped warbler (<i>Dendroica coronata</i>)	-58	-43
Ruby-crowned kinglet (<i>Regulus calendula</i>)	-31	-10
Magnolia warbler (<i>Dendroica magnolia</i>)	-20	-19
Ovenbird (<i>Seiurus aurocapillus</i>)	-10	-6
Bay-breasted warbler (<i>Dendroica castanea</i>)	+5	+32
American redstart (<i>Setophaga ruticilla</i>)	+24	+87
Tennessee warbler (<i>Vermivora peregrina</i>)	+83	+107

* Data are percentage changes in bird numbers after treatment of 420 g/ha of fenitrothion to Canadian forests. Source: Peakall and Bart (1983).

control programs, swaths are applied across only relatively small areas occupied by bands or swarms as they are flying, resting, or feeding. In Africa, control of bird pests, such as red-billed quelea (*Quelea quelea*), is conducted at dusk just after birds have arrived in breeding colonies or roosts. The airplane flushes the birds, which then circle and twist in a compact, tight band over the small area of the colony or roost, where they are sprayed with a dermal-contact avicide. Queletox, the avicide most commonly used in Africa, is applied at relatively high rates in a ULV formulation. Tens of thousands of quelea may be killed, but the mortality of nontarget birds is usually rather low. In 2 breeding colonies and 2 roosts in Kenya, from 10-100 L of Queletox (600 g/L of fenitrothion) were sprayed over the birds. Based on the surface area of the roosts and colonies, these applications would compare to applications of 1.5-12.0 kg/ha of conventional swath treatments; amounts actually applied over birds were, of course, even higher. Only 22-61 dead, nontarget birds were found following these four Queletox applications (Bruggers et al. 1989, Keith et al. 1994). In contrast, hundreds of dead birds have been found in areas treated at much lower dosages for mosquito control in the United States. De Weese et al. (1983) found 99 dead, nontarget birds following fenitrothion applications of 0.045-0.056 kg/ha to flooded pastures in Wyoming. In North Dakota, mosquito control in woodlands with 0.09 kg/ha of fenitrothion killed thousands of nontarget birds; 453 dead birds were picked up from only a small portion of the treated area (Seabloom et al. 1973).

Can fenitrothion applications of 1.5-12.0 kg/ha kill fewer nontarget birds than those of 0.045-0.090 kg/ha? The answer is yes, and the reason is that heavy doses of fenitrothion are applied to very small areas (10s ha) in quelea control and almost every nontarget individual that is present under the plane is killed. Conversely, application rates for mosquito control, which are adequate to

kill birds, may cause death to many more birds over the much larger areas that are treated (100s-1000s ha).

Variation in effects among environments

During the recent irruption of desert locusts in Africa, fenitrothion was used against the massive swarms as they moved across the Sahel. However, studies had not been conducted to evaluate effects of fenitrothion in the Sahelian environment. Rates of fenitrothion applied were about 500 g/ha. There was great concern about the use of fenitrothion because of its reputation as a bird killer. Several studies showed that fenitrothion killed birds or reduced their activity (Smith 1987, McEwen 1982, Grant 1989, Crick 1990).

Mullié and Keith (1993) evaluated fenitrothion effects on birds in semiarid thornbush savannah of northern Senegal. Tree and shrub canopy cover ranged from 10-20%, and 57% of that cover was less than 2.5 m in height. ULV applications of fenitrothion at 485 and 825 g/ha were applied. Following treatments, native herdsman located only a few dead birds, an average of 0.07 birds per man-hour of search. Search efficiency and carcass disappearance rates were measured to enable calculations of population mortality (Table 4). Confidence in search efficiency was supported by the recovery of numerous flightless fledglings of five species. Numbers of the 21 most abundant species seen during bird counts decreased after treatment (-46 and -63% on plots treated with 485 and 825 g/ha, respectively), apparently due to birds leaving treated areas. In Canada, sick and dead birds were found after fenitrothion applications of 280 g/ha (Peakall and Bart 1983). In studies of 420 g/ha applications, from 1 to 6.8 sick birds were found per man-hour of searching (Pearce 1968). At the same application rate, Bart and Hunter (1978) recovered up to 4.5 sick or dead birds per

Table 4. Birds found dead and calculated population mortality after fenitrothion application in Senegal.*

	Application rates (g/ha)	
Bird mortality	485	825
Birds found dead (#)	3	10
Population mortality	4.5	6.5

*Source: Mullié and Keith (1993).

man-hour of searching. Buckner and McLeod (1977) reported temporary declines in bird activity at 210 g/ha, mortality of nestlings and fledglings at 210 to 280 g/ha, and adult mortality at rates above 280 g/ha.

At 1 week post-treatment in Senegal, ChE inhibition in live birds averaged 27 and 42% on plots treated with 485 and 825 g/ha of fenitrothion, respectively. Busby et al. (1983) found average ChE inhibition of 20% at 210 g/ha and of 42% at 420 g/ha in live birds 3 days after fenitrothion applications in New Brunswick. In two studies, inhibition averaged 29% in Scottish birds during the first week after fenitrothion treatments of 300 g/ha (Hamilton et al. 1981, Spray et al. 1987). ChE inhibition, as mortality, was lower after 485 and 825 g/ha applications in Senegal than following applications at rates of one-half that amount in Canada and Scotland.

What factors could have been responsible for birds being more severely affected by fenitrothion in Canada than in Senegal? Likewise, with the limited knowledge of fenitrothion behaviour and ecological conditions in the two countries, could differences in effects have been predicted? There is the possibility that the species of birds in Canada and Senegal differed in their susceptibility to fenitrothion. Considerable variations are known to exist in the toxicity of fenitrothion to birds (Hudson et al. 1984). There is little to suggest fenitrothion residues were greater or persisted longer in Canada. Residues of fenitrothion decreased rapidly after applications in both countries.

On fir foliage, 50% was lost in 4 days in Canada (Yule and Duffy 1972), while on poplar and birch, residues decreased from about 20 ppm to about 1.0 ppm in 10 days (La Pierre 1985). In Senegal, 70 to 90% of initial residues on grasses disappeared after 4 days (Everts 1990).

Spray deposits, their persistence, and their effects can be related to droplet size (Keith 1968). Pearce (1968) reported only 0.6 and 0.3 sick birds per man-hours of search on plots treated with coarse droplets of fenitrothion at 420 and 560 g/ha, respectively. Greater numbers of sick birds (1.0 to 6.8 birds per man-hour) were found on plots sprayed with fine droplets at 420 g/ha. It also has been documented that meteorological conditions (wind, temperature, relative humidity, and thermal action from the warming of ground surfaces by the sun) can influence spray deposits. Certainly, the high temperatures, minimal vegetative cover, and greater thermal activity in Senegal would be expected to restrict deposits more than conditions in the cooler forests of Canada.

Vegetative cover as well as the habits and behaviour of birds in treated areas can influence the intensity of their exposure. In Canada, the impact of fenitrothion applications on birds feeding in the upper canopy of the forest were greater than on ground-feeding species (Peakall and Bart 1983). Mullié and Keith (1993) found that decreases in bird abundance following fenitrothion applications varied with habitat preference, feeding strategy, and diet of birds. In Senegal, the open thornbush savannah may have enabled birds to more rapidly flush from the path of the spray aircraft and escape heavy direct dermal exposure.

Several studies have shown that the greatest exposure of birds to insecticides results from dermal absorption at the time of spraying (Hamilton et al. 1981 Findlay et al. 1974). Ingenious laboratory investigations by Driver et al. (1991) showed the relative

contribution of various routes of parathion exposure to birds. By isolating routes of exposure and measuring ChE inhibition and parathion residues, the investigators showed the most important routes of exposure to be inhalation and dermal at 1 hr after application, ingestion after 4 hr, and dermal absorption at 8, 24, and 48 hr (Table 5). Differences between Senegal and Canada in fenitrothion effects on birds could have been due to variations in dermal exposure of birds.

Conclusions

Given the variations in effects of insecticides on wildlife that are discussed here, it is difficult to imagine how any set of desk-top appraisals can hope to identify the potential hazards of insecticides before they are registered for field use. How can we anticipate and incorporate in our decision-making processes such factors as: 1) differences in residue persistence related to habitat characteristics; 2) differences in the fate and effects of insecticides among habitats, such as the Oregon lakes or Canada and Senegal; 3) differences in insecticide deposits and the contamination of treated habitats due to meteorological conditions and the mechanics and strategies of spray applications; 4) differences in the exposure of animal species due to their habits and behaviour; 5) differences in the toxicity of insecticides to the various kinds of wildlife on treated areas; 6) unexpected effects such as eggshell thinning and indirect effects that can result from pesticide use.

Smith (1987:5) agrees that "extrapolation from laboratory studies to the field is usually approximate at best and sometimes not possible." Kendall and Akerman (1992:1727) recognized that "addressing the ecological risks associated with the use of an agricultural chemical involves a complex array of laboratory and field studies, in essence, a research program."

Laboratory data such as the relationship of chemical structure to activity have commonly been used in attempts to estimate the field hazard of insecticides (Kenaga 1973 and 1982, USEPA 1978). These analyses have provided additional information that has been useful in assessing relative environmental hazards of new insecticides. However, the assumptions made in these models require that environmental factors remain constant; and as we have seen, most often field conditions tend to be highly variable. Mackay (1993) claims to have developed a procedure for determining the fate of chemicals over broad political areas or ecological regions. I believe his basic assumption of the continuity of conditions over large areas is invalid. For example, the fate and effects of toxaphene differed dramatically between two lakes in a single ecological region, the Oregon Cascades.

The concept of developing predictive models to identify hazardous pesticides is an enticing one because the process would allow us to eliminate problems before they occur. However, the exciting aspect of pesticide-wildlife research has been the continued

Table 5. Percentage of brain ChE inhibition due to different routes of parathion exposure.*

Route	Hours post-spray				
	1	4	8	24	48
Inhalation	17	8	6	<3	<3
Dermal	16	10	18	28	42
Food ingestion	-	14	11	20	10
Preening ingestion	8	16	10	17	8
Total inhibition	41	48	45	68	63

* Source: Driver et al. (1991).

confrontation with new and unexpected problems. Complexity is inherent in ecological relationships and predictability is the exception. Such experience and knowledge does not suggest that prediction of pesticide hazards can often be accomplished.

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